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BENIGN PLASMA-CELL ERYTHROPLASIA.

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SINCE Queyrat's article (1911) the term erythroplasia has been applied to lesions of the genital and buccal mucous membrane characterized by red shining patches with a varnished appearance, painless and, according to most authors, developing sooner or later into epitheliomas. Darier identified erythroplasia with the benign syphiloid epithelioma which he had described with A. Fournier.

It is generally accepted (as confirmed by the majority of recent texts on dermatology) that erythroplasia is a pre-cancerous state, the diagnosis of which implies a guarded prognosis and indicates destructive treatment, either surgical or radio-therapeutic. For many years various publications on the subject, particularly in France but also in other countries, have supported this concept, and in 1936 Touraine and Solente wrote: "long classified among the pre-cancerous states, erythroplasia is to-day regarded as a true cancer".

However, certain reports of clinical erythroplasia have noted the absence of any histological evidence of malignancy. Touraine and Solente, who collected 92 published cases, point out that in 57 there was no mention of malignant degeneration. To explain this inconsistency two histological types of erythroplasia are distinguished: one with simple hyperplasia without cytological abnormalities and the other with metaplasia and unquestionable malignant changes. In 1940 I published the report of a balano-preputial lesion which presented diagnostic difficulties, with all the clinical features of erythroplasia but which, after a duration of 5 years, showed no sign of malignancy on histological examination. Other observers have mentioned a benign course. Louste, Levy-Frankel and Cailliau published, in 1931, a case of erythroplasia cured by arsenical ionization. In 1933 Sulzberger and Satenstein reported the first American case of erythroplasia of the glans without

histological evidence of malignancy. During a period of more than 20 years under observation this case had never shown malignant change. In 1948 W. Sachs and P. Sachs reported, under the designation of erythroplasia of Queyrat, lesions of the genital mucous membrane and of the skin in Jewish patients in whom histological examination showed no malignant change but an infiltrate of plasma cells. They concluded that erythroplasia is not a cancerous or pre-cancerous condition but a curable inflammatory disorder.

It was Zoon who, in 1952, in reporting 8 cases of chronic circumscribed plasma-cell balanoposthitis crystallized the position by insisting on the benign nature of the condition, which he differentiated from Queyrat's erythroplasia and which is characterized by an infiltrate of plasma cells. This infiltrate of plasma cells had, in fact, already been noted by various authors and by Negri (1932) in particular. Faithful to the classical theory of the malignancy of erythroplasia, he thought that this infiltrate was one of the diagnostic features of pre-cancerous states. Credit must therefore be given to Zoon for having made this histological sign the distinctive feature of the benign nature of the condition.

Since then several authors have supported this point of view, among them Roederer (1953) in the report of a case of plasma-cell balanitis. In 1954 I reported two cases of benign chronic erythematous plasma-cell vulvitis, the counterpart in women of the lesions reported by Zoon. Subsequently Graciansky, Boule and Grupper (1954), Duperrat and Goetschel (1955) in France, Nödl (1954), Nikolowski and Wiehl (1956) in Germany, accepted this concept of plasma-cell balanitis. Quite recently S. Blau and A. B. Hyman (1956) have recognized three types of erythroplasia: Bowen's disease, a non-specific inflammatory reaction and plasma-cell infiltration.

I would like in this short paper to present an account of the clinical aspects of these plasma-cell erythroplasias, and of their histology, and finally to discuss their nosological status.

CLINICAL ASPECTS.

These lesions can appear at various ages: one of Zoon's patients was 24, another was 74. My two patients with erythematous plasma-cell vulvitis were young women aged 31 and 28 years. Malignant erythroplasia usually affects elderly subjects, but benign erythroplasia can occur in the young. The lesions are most commonly situated on the genital mucous membrane, and usually involve the mucous membrane of the glans and prepuce. Reports of benign erythroplasia of the vulva are much less common; I have found no reports other than those I published in 1954, though there must evidently have been some erroneously classified as malignant erythroplasia. A case involving the buccal mucous membrane has been reported by Nikolowsky and Wiehl.

The lesions correspond more or less closely to the classic descriptions accepted since Queyrat's day: they are bright or dull red patches of the mucous membrane, with a glistening varnished appearance, which in several descriptions has been likened to lacquer. In my two patients with erythematous vulvitis the patches had an ecchymotic tint which was quite distinctive and the explanation of which we shall later see provided by the histology. It is to be noted that this ecchymotic red colour is also recognized in men and that in certain reports a red-brown chocolate tint is described. Thus it was in the patient which I reported in 1940 and in Duperrat and Goetschel's patient.

A clinical feature which I consider important, and which was present in my two patients, is the irregular indefinite and ragged outline of the patches in contrast with malignant erythroplasia in which the outline is customarily sharp.

The patches on the penis are usually painless but one of my patients with vulvitis complained of tenderness and the second of pruritus. On examination the lesions are soft or slightly infiltrated but are not significantly raised above the level of the healthy mucous membrane.

The course is chronic and may extend to many years—from 8 years in one of my patients to 12, 13 and 20 years in certain of Zoon's patients, and this without the least malignant change and without adenopathy. This chronic course is not modified by treatment. I well recall that in my first patient, in whom erythroplasia had been present for many years, I electrocoagulated the lesions because, in spite of the absence of any evidence of malignancy in the biopsy, the cyto-diagnostic test had been declared suspect. As a sequel to this operation identical patches reappeared after cicatrization. Zoon, in his 8 cases, only once secured the disappearance of balano-posthitis and this was after contact radiotherapy of 3000 r.

HISTOLOGY.

It is particularly the histological examination which permits the recognition of benign erythroplasia; clinical examination provides only presumptive signs and caution requires the exclusion of malignant change.

The epithelium shows either a moderate degree of acanthosis, as in my two patients, or an atrophic appearance (7 of Zoon's 8 cases), or a combination of acanthosis and atrophy. Blau and Hyman state that the acanthosis does not involve the supra-papillary regions where the epithelium is normally thin and the vessels are near the surface. Almost all authors have noted a clear appearance in the epidermal cells, the result of intra- and extra-cellular oedema. This oedema gives the cells an hydropic appearance which makes them resemble dyskeratotic cells, and which has led to misinterpretation by certain authors; it is I think the explanation of the error in the interpretation of the cyto-diagnostic test in my first patient. In contrast, there are no alterations in the

general architecture of the epidermis, and none of the irregularities and nuclear anomalies which are the rule in the malignant erythroplasias of Bowen's type. The basal layer remains normal.

It is in the dermis that one finds the distinctive feature of benign erythroplasia: the inflammatory infiltrate concentrated in the corium and formed of plasma cells. It is often an almost pure infiltrate containing only plasma cells or plasma cells with a few lymphocytes and eosinophilic polymorphonuclears. Several authors note the presence of Russell bodies which are derived from plasma cells (Zoon; Nikolowski and Wiehl). Beneath this infiltrate there is sometimes a band of normal connective tissue. Important vascular changes are associated with this plasma-cell infiltrate: the capillaries are dilated and their walls are thickened, the combination recalling the pseudo-syphilitic plasmoma (Roederer; Garnier). Finally one finds blood pigment fairly abundantly and these deposits of haemosiderin, demonstrated by Prussian blue staining, are an important histological feature which accounts, I believe, for the ecchymotic or red-brown chocolate colour of the lesion which is so distinctive (Garnier; Duperrat and Goetschel).

This histological picture of benign erythroplasia partly resembles a description which has been given under the name of a hyperplastic form of Queyrat's erythroplasia; one knows however that a large number of the latter have never shown signs of malignancy and one may surmise that some at least were really benign plasma-cell erythroplasia.

DIAGNOSIS.

Benign erythroplasia, as indeed the malignant form, must be distinguished from persistent red patches of the genital mucous membrane in psoriasis, lichen planus and eczema, from diabetic vulvitis or balanitis, recurrent inflammatory balanitis of the sixth decade, and from the erythematous vulvitis of menopausal or ovariectomized women. American authors insist on the differential diagnosis from the lesions of the exudative discoid and lichenoid dermatosis of Sulzberger and Garbe, which is recognized by its prolonged course, by its evolution in four eruptive stages (exudative, lichenoid, infiltrative and urticarial) and by other cutaneous lesions. What complicates the problem, at least for dermatologists in the United States where this dermatosis is encountered, is that the histological examination shows a plasma-cell infiltrate which may lead to great difficulty if there happens to be only the lesion on the glans and the skin lesions in other sites have disappeared. This condition, which affects Jews almost exclusively, probably corresponds to the cases described by W. Sachs and P. Sachs under the name of erythroplasia of Queyrat and which were cured by local applications of arsphenamine.

The plasma-cell infiltrate associated with dilated capillaries with thickened

walls has suggested a possible syphilitic aetiology. Queyrat emphasized the frequency of syphilis among his patients with malignant erythroplasia, but this has not been confirmed by other reports. As far as benign erythroplasia is concerned nothing permits one to accept this aetiology and one can merely talk of a probable inflammatory cause but at present, still non-specific.

NOSOLOGY.

What nosological status ought one to accord to benign plasma-cell erythroplasia? Should one, like certain authors, consider that it comprises all erythroplasia? I think not; one must, in my opinion, accept: (1) Malignant erythroplasia (Queyrat) which is recognized by its distinctive histological features and is really a variant of Bowen's disease. It requires the vigorous therapeutic approach of any cancerous lesion. (ii) Benign plasma-cell erythroplasia where the histological examination allows the pessimistic prognosis and mutilating treatment to be avoided.

This new concept of a benign erythroplasia deserves emphasis, I believe, both because of its undoubted practical importance and because from the theoretical point of view it allows the classic theory of the inevitable malignancy of certain lesions to be corrected.

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PORPHYRIA CUTANEA TARDA FOLLOWING JAUNDICE.

REPORT OF TWO CASES.

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THERE is a steadily-growing list of reported cases of porphyria in which the main or the only clinical feature has been a bullous dermatosis. The two cases presently reported have the additional interest of having both followed an attack of jaundice. This association of porphyria and jaundice has been referred to previously. Brunsting and Mason (1946) described a case of "epidermolysis bullosa" with hepatic dysfunction and familial porphyria. The same authors (1947) described four further cases of epidermolysis bullosa-like eruptions associated with porphyria in which the skin syndrome was precipitated by liver damage due to disease, hepatotoxic drugs or poisons. Gray *et al.* (1948) give an account of a case of porphyria associated with recurrent attacks of jaundice and point out that the regular excess of coproporphyrin I (and probably III) is excreted by the liver and found in the stool, but that, during jaundice, it is diverted to the blood stream (producing the photosensitivity and colic) and thence to the kidneys. They suggest that an ether soluble porphyrin may produce the hepatic dysfunction and consequent diversion of coproporphyrins. This latter theory supports the view of Turner and Obermayer (1938) that the hepatic dysfunction is caused by the disordered porphyrin metabolism and does not play any part in its genesis.

Although the bullous eruptions occurring in cases of porphyria cutanea tarda have often been described as epidermolysis bullosa, important differences are pointed out by a number of authors (Winer and Orman, 1945; Blum, 1941; Brunsting and Mason, 1949; Turner and Obermayer, 1938). The principal precipitating factor in epidermolysis bullosa is trauma, and the sites affected are those exposed to pressure and friction but not necessarily to light, as for example, the soles. In porphyria, the bullae occur only on light exposed surfaces though often in response to trauma.

CASE REPORTS.

CASE 1.—A radio engineer, aged 22, serving at the time in the Royal Air Force.

At the beginning of August 1954 he developed jaundice with malaise, fever and dark urine. A few days later vesicles appeared on the face, followed within 2-3 days by bullae on the dorsal surfaces of the hands and forearms. The jaundice cleared in

2-3 weeks but further bullae continued to appear and, on 29 August 1954, he was admitted to hospital. During the period of six weeks in hospital, the bullae gradually lessened in number and severity and this improvement continued after his discharge. However, a year later, a few bullae were still appearing on his hands from time to time, often in response to trauma.

As a child he had recurrent attacks of abdominal pain, but never had jaundice before. Since childhood he had noticed that trauma to the skin on his hands readily resulted in separation of the upper layer of the damaged skin. There is no history of mental disorder or alcoholism. No drugs had been taken prior to the onset of the jaundice. His mother had jaundice in her youth but otherwise there was nothing relevant in his family history. His parents were not related.

Once the jaundice was clear examination revealed no abnormality other than vesicles and bullae scattered over the face, neck and backs of hands and fingers. Where the lesions had healed the area became pigmented and numerous epidermic cysts developed. While he was an in-patient various tests were made to determine sensitivity to ultra-violet rays and trauma. Exposure of the forearm and hand to mercury vapour lamp, carbon arc lamp and natural sunlight produced normal results. Pinching of the skin with forceps resulted in the formation of bullae only in an area normally exposed to light, for example, on the dorsum of the hand, but not on the abdomen or leg.

The following is a summary of the laboratory investigations :—

Haemoglobin, 97% ; W.B.C., 6700.

Liver function tests :—

	1.ix.54.	5.x.54.
V. den Burgh (indirect)	0.7 mg. %	0.3 mg. %
Serum alk. phosphatase	8 units %	5 units %
Serum proteins	7.0 g. %	6.9 g. %
Serum albumin	4.5 g. %	4.0 g. %
Serum globulin	2.5 g. %	2.9 g. %
Thymol turbidity	10 units	3 units
Thymol flocculation	Complete	0
Colloidal gold flocculation	2 units	0

Urine :

27.viii.54	Fouchet's test for bile pigment	Negative.
27.viii.54	Waldenstrom's test for porphyrin	"
1.ix.54	Bilirubin	"
1.ix.54	Urobilin	Positive.
1.ix.54	Urobilinogin	"
1.ix.54	Porphyrins	Negative.
1.ix.54	Porphobilinogen	"

Stool :

1.ix.54	Porphyrins ++ (qualitative only).
15.iii.55	Porphyrins (Professor Rimington).
	Urine Porphyrins 390 mg./g. (normal 0-200)
	Stool Coproporphyrin 509 mg./g. dry weight (normal up to 15)
	Stool Protoporphyrin 792.7 mg./g. dry weight (normal up to 30).

CASE 2.—This patient was first seen in 1952, when he was a medical student.

In April 1940, when 16 years old, he developed jaundice which was diagnosed as infective hepatitis and followed the course of a typical attack of that disease. During the attack he suffered upper and central abdominal pain and constipation. Five

weeks after the onset, when the jaundice had cleared, on going out into the sunshine, itching developed on the hands, face and neck. This was followed 24 hours later by considerable oedema of the face and hands and the appearance of bullae on the exposed skin. The oedema disappeared in 36 hours but the bullae persisted. During the next 6 years bullae continued to appear throughout the year but always worse in summer. This tendency has gradually cleared although the skin is still easily damaged by trauma. There is no history of mental disorder, alcoholism or previous jaundice. Since childhood the superficial layers of the skin of his hands have been readily knocked off by trauma. His brother, his father and one of two paternal uncles have all been afflicted with the same easily-damaged skin on exposed areas, but no illnesses are known in his own or in his father's generation which suggest attacks of acute porphyria. His paternal grandmother died from pernicious anaemia. His parents were not related.

The following investigation was carried out in April 1955 (Professor Rimington) :—

Urine : Porphobilinogen test : Faintly positive ($\pm$).

Stool :

Coproporphyrin 196.7 mg./g. dry weight (normal up to 15).

Protoporphyrin 426.0 mg./g. dry weight (normal up to 30).

DISCUSSION

Both patients gave a history of having an easily-damaged skin since childhood. The bullous eruption following jaundice gradually cleared and, after a period of six years in Case 2, the skin had returned to the same easily-damaged state as before. In Case 1 the skin appeared to be gradually approaching the pre-jaundice state a year after the attack. However; at this stage in both cases grossly abnormal porphyrin excretion was demonstrated and it seems likely that the abnormal porphyrin metabolism had been present since birth.

The attacks of jaundice clinically resemble infective hepatitis. No hepatotoxic drugs or excess of alcohol had been taken and the jaundice was not recurrent. If these attacks of jaundice were caused by the altered porphyrin metabolism, as has been suggested in other reported cases, it is surprising that similar attacks have not happened before or since. There is also an interval of five weeks in Case 2 and a few days in Case 1 between the jaundice and onset of the eruption. The most likely explanation in these cases seems to be that liver damage due to infective hepatitis led to an alteration of the already disordered porphyrin metabolism and the production of a high level of porphyrin in the blood. If this is so, it is difficult to explain the cases of porphyria in which recurrent attacks of jaundice have been described. It seems possible that in patients with porphyria the liver is more readily damaged by hepatotoxins or the virus of infective hepatitis.

The history of easily-damaged skin ; the family history of such in Case 2 ; the production of blisters by trauma ; and the development of numerous epidermic cysts on the sites of healed blisters as Case 1—are all commonly present in epidermolysis bullosa. The main difference in porphyria is that the bullae follow trauma to exposed surfaces only. However, these similar

features suggest the possibility that there may be some biochemical or genetic relationship between porphyria cutanea tarda and epidermolysis bullosa.

SUMMARY

A brief résumé is given of the literature concerning porphyria with bullous eruptions, with particular reference to the association with jaundice. Attention is drawn to the distinction between these eruptions and epidermolysis bullosa.

Two cases are described of porphyria and bullous dermatoses following jaundice.

The problem of the causal relationship between hepatic damage and porphyria is discussed.

We are indebted to Dr. C. D. Evans for his helpful co-operation in the investigation of these cases.

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AN EFFICIENT TREATMENT FOR ACNE VULGARIS.

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A PERUSAL of the literature indicates that the majority of dermatologists have no standard efficient treatment for acne vulgaris, nor do the experiences of different dermatologists with any given treatment seem to be the same. For example, Kline (1954) says "vitamin A therapy is of special value in treatment of acne vulgaris" while Osbourn (1954) writes "vitamin A is without value in treatment of acne vulgaris". In 1954 three current journals carried articles mentioning, in all, about 45 preparations or procedures (including removal of tonsils) as having merit.

It is extremely desirable that we should be able to "cure" acne vulgaris. Osborne (1947) described it as "the commonest disease process known to man . . . a disease that probably caused more man-hours lost during the war than almost any other condition". He said "it is estimated by competent authorities that at least 50% of young people between the ages of 17 and 25 manifest some degree of acne vulgaris". Rothman (1955) suggests acne vulgaris is foremost among many pathological conditions that "never endanger but often ruin life".

There seems to be a considerable volume of opinion that the two principal causes of acne vulgaris are a hormone imbalance and bacterial infection. Cronk *et al.* (1956) speak of "the hormonal-bacterial etiology of acne" giving as references articles by Becker and O'Brien. Sullivan and Zeligman (1956) discussed the role of hormones and also of bacteria in causing acne vulgaris; and in discussing their paper Rothman gave a reasonable explanation of this dual aetiology when he said "when sebum stagnates inside the follicle its decomposition leads to formation of products which seem to be a good culture medium for bacteria".

Over a considerable number of years the author has treated several hundreds of cases of acne vulgaris by a method which has given very close to 100% clearing up of all cases. There seems to have been a minimum of scarring. This may be due to the fact that the treatment is essentially what one might call physiological, depending upon adequate treatment of the two causes of acne *at the same time*. This method of treatment has been reported briefly in the past (Baird, 1949) without much explanation but seems to have been taken up by only a few dermatologists, although a number of general practitioners have privately reported excellent results. This paper is intended to explain

the rationale of the treatment, and to remove some misconceptions from the minds of physicians who will not even attempt to use it because of certain *theoretical* objections.

In brief, the treatment consists of altering the hormonal imbalance to a more normal state by the use of chorionic gonadotropin and raising the resistance of the patient's blood serum against bacterial infection by means of adequate doses of bacterial antigen-antibody.

As has often been pointed out, acne shows a tendency to spontaneous remission so that it is difficult to evaluate treatment. The following facts seem to indicate that the clearing in the author's cases is due to the treatment, and not merely coincidental :

In cases of long standing and in cases of only a few weeks' duration improvement is regularly shown soon after beginning the treatment.

In a number of cases where the treatment has been interrupted the improvement has ceased.

In the small number of cases where the acne has recurred the relapse has promptly receded with a small amount of further treatment.

The hormone treatment can be used alone very successfully to control those cases of oily or greasy skin or which have a tendency to develop sebaceous cysts, but where the bacterial element does not seem to be present and there are no inflammatory papules or pustules.

The vaccine therapy as described is extremely efficient in various non-acne pyodermas.

In acne, where both glandular and bacterial elements exist as causes, the use of either one of these treatments by itself will not result in improvement in a very large percentage of cases. They must be used together.

Correction of the endocrine imbalance with oestrogens is so well known that no attempt will be made here to review the bibliography on this subject. There have been some reports of good results with the local use of oestrogen preparations. On theoretical grounds the author believes that it is more reasonable to use stimulation therapy. It may be assumed that in cases which subside spontaneously the improvement takes place because the endocrine balance from the patient's own glands has become normal. One would think that if this can be caused to take place by means of treatment the results would be much more likely to be permanent, and the use of gonadotropic hormone appears to do this with great regularity.

The author has heard a considerable number of clinicians express fear that use of gonadotropic hormone would somehow *upset* the patient's glandular functions. This fear can only result from a failure to realize that the patient already has an upset or imbalanced situation. This seems to be restored to balance by the treatment recommended, and in hundreds of cases over many years the author has not yet seen anything to justify the timidity of those who

are afraid to use it because of a fancied danger of disturbing menstruation, causing enlargement of breast tissue in the male, and other endocrine effects. On the contrary one can assure girls and young women who suffer a great deal of pain during menstruation that this disabling condition will in all probability disappear along with the acne.

Concerning the infective element in acne Robinson (1954) rightly says "the role of pyogenic bacteria in the production of acne pustules has evoked considerable discussion among dermatologists, and there is a wide divergence of opinion concerning its importance". The situation calls for some clear and logical reasoning based upon what is already known about the body's reaction to the presence of bacteria. For example, when Robinson points out that local antibiotic therapy in acne is not advisable because of too few satisfactory results and the danger of producing contact sensitivity, he is reporting not only results of his own experience but also what one would reasonably expect. If the underlying trouble is a relative lack of those materials in the blood which produce resistance to bacteria, then it is not likely that reducing the number of bacteria on the surface of the skin for a time will produce any permanent effect. It is well known that antibiotics do not increase the patient's resistance but in some cases actually decrease it by removing the stimulation which the presence of bacteria normally provide. Moreover if, as Rothman suggested, decomposed sebum in the follicle is a good culture medium it is not likely that antibiotics will do much to change this.

Similar reasoning would apply to the systemic use of antibiotics to which Robinson attributes some value as supplementary treatment.

The best, safest, and longest lasting "antibiotic" is found in those substances which the body elaborates to protect itself against the presence of bacteria. Their action, though specific against the bacteria which have stimulated their formation in a given case, has many side reactions (Wiener, 1956). In other words it is not always necessary to use the exact bacteria concerned in order to stimulate a helpful non-specific reaction to other organisms (Wright, 1944). The question is then whether or not it is possible to stimulate sufficient resistance on the part of the patient to clear up the given infection. Many attempts have been made to do this with bacterial vaccines. As a result of individual experiences dogmatic statements have often been made that vaccine is of little or no use in the treatment of acne. For example, concerning the value of vaccines in acne, Osborne (1947) states "that, of course, has been exploded in our literature. Vaccines are of no value in acne vulgaris". In 1952 in a discussion of acne in *Physicians' Bulletin** vaccines are listed with a number of other treatments that "apparently have little value in acne". Borrie (1956) stated recently concerning vaccines, "this method of treatment for chronic or recurrent staphylococcal infection is of doubtful value" but added that if it is to be used

* Published by Eli Lilly & Co., 1952, vol. 17, 4 : 57.

in acne "an autogenous vaccine must be used". (The statement, "an autogenous vaccine must be used" is his private opinion, but he has already stated that vaccines are of doubtful value. Therefore why *must* one use an autogenous vaccine which one may assume he finds to be "of doubtful value"?)

Many similar statements have been made by writers who seem to think they have proved a negative. All they have proved is that the particular vaccines *they* were using, in the dosages and by the methods *they* describe, failed to produce satisfactory results, but this does not permit them to generalize on the subject of vaccine therapy. Such generalizations are often made and accepted by medical editors, thereby doing a great dis-service to progress (Baird, 1955).

It is a clinical fact, which can be verified by tests, that by giving bacterial antigen-plus-antibody in sufficiently large doses it is possible so to alter the patient's resistance against ordinary skin-infecting bacteria as to stop recurrent attacks of pyoderma. In acne vulgaris the resistance becomes so high that pustulation decreases and as soon as the secretion and excretion of sebum has become more normal it ceases altogether. These statements should not cause the reader to protest that vaccine therapy has already been proved to be useless in acne, but rather to inquire wherein lies the difference in product, or in technique of administration, between the treatment here described and the previous uses of vaccine which did not produce such results. Any other experience with vaccine therapy is not likely to have much bearing upon the above statements, particularly since the doses recommended are so much larger in terms of killed bacteria than those employed in any treatments the author has been able to find described in the literature.

The author's method of treating acne vulgaris based upon the preceding principles is as follows. Intramuscular injections of at least 500 international units of chorionic gonadotropin are given about once a week in the male. The same dosage has been used with success in young women who have no clues as to when they ovulate because of having had an early hysterectomy. In other young women and girls the doses of chorionic gonadotropin are usually 500 international units about one week and three weeks after the first day of menstruation, and 1000 international units about two weeks after the first day of menstruation. In cases which are nearly cured it is often wise to continue the dose of 1000 international units once a month for a few more times.

In all of the above types there is an occasional case which does not seem to be showing any improvement after several weeks of treatment. These cases will usually start to improve when the doses are increased by about 50%.

Sensitized mixed vaccine ("Serobacterin") is given to increase antibacterial resistance. The doses used are much higher than those recommended by the makers. The author usually employs Staphylo "Serobacterin" Vaccine Mixed*

* Messrs. Sharp & Dohme.

but if a particular patient has a great deal of upper respiratory trouble *H. influenzae* "Serobacterin" Vaccine Mixed* may be used to clear up this condition, and as a rule it is sufficient as treatment for the acne also. The question may be asked why these mixed vaccines are used rather than one limited entirely to staphylococci. A partial answer to this is that there is increased evidence of cross-action of antibodies or what one might call the non-specific immunizing effects of vaccines in general. This principle was emphasized over thirty years ago by Wright (1944) and in 1956 by Wiener. The vaccine should be given *subcutaneously* and the usual doses are :

0.2 c.c.; 0.4 c.c.; 0.8 c.c.; 1.2 c.c.; 1.8 c.c.; 2.5 c.c.

These doses may be given at intervals of four or five days but it is usually quite suitable to give them when the patient comes in for the weekly injection of gonadotropin. The 2.5 c.c. dose may need to be repeated a number of times at intervals of two to four weeks. Occasionally doses as high as 3.0 c.c. or 3.5 c.c. are required. Amounts of over 1.0 c.c. are usually given one-half in each of two places. Should there be a particularly severe local or general reaction—which is rare—the dose which caused it can usually be repeated a week later with no particular difficulty.

As with all skin reactions, in addition to removing the general cause it is well to treat the local symptoms. In the case of acne this is well accomplished by washing twice a day with warm soapy water, rinsing, and douching with cold water, followed by the application of a sulphur lotion. The only dietary restriction the author generally advises is the avoidance of all chocolate products. This is based on the observation that a number of patients develop acne-like eruptions within a few hours of eating chocolate, and the idea that chocolate contains traces of bromine.

SUMMARY.

Treating endocrine imbalance and low antibacterial resistance synchronously is stated by the author to have cleared up nearly all of several hundred cases of acne vulgaris.

Stimulation with gonadotropic hormone is used in preference to mere substitution therapy. Not only has it not produced disturbances of glandular function, but it seems to have corrected imbalances not related to the acne, such as painful menstruation.

An antigen-antibody preparation is used in very large doses to increase resistance against bacteria. Other experiences with much smaller doses of bacterial antigens alone must not be used as basis for theoretical criticism of this treatment. Its value can be proved or disproved only by test, not argument.

* Messrs. Sharp & Dohme.

Neither the hormone treatment nor the vaccine treatment are of great value in acne vulgaris when used alone. It is their use *at the same time* which is recommended.

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DOGGER BANK ITCH.

REPORT OF A CASE

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CONTACT dermatitis due to the Coraline *Alcyonidium hirsutum*, the "sea chervil", is sufficiently common in countries adjoining the North Sea for it to have been recognized in the Danish Workman's Compensation Act since 1939 (Bonnevie, 1948). It is rare in North Lancashire and only one case has been seen in three-and-a-half years.

The patient, a sea captain, first attended in 1953. He was of Norse extraction and had been born in Iceland. There was no past or family history of allergic disorders.

Most of his 42 years at sea were spent near the Dogger Bank and he says the sea chervil, which he calls "curly weed", is abundant there and comes up with the nets. It has to be thrown back into the sea. Since the war the "weed" has become more widespread in the North Sea; he has also found it in Luce Bay, Wigtonshire.

In November, 1934, when fishing off the North Dogger, probably with his jersey sleeves rolled up, he developed an itching red eruption on the flexor aspects of the elbows and forearms which became moist, oozed serum, and spread to involve the backs of the hands, fingers and most of the arms within a few days. He said that his arms became swollen to one and a half times their normal size and that this was a feature of the illness recognized by fishermen. The rash started to clear when he left the area and after some days the skin appeared normal.

His next attack was on going to the same area again, and this occurred repeatedly, the duration of the attacks lengthening. In 1938 the face and neck became involved, with puffiness of the skin around the eyes; he tells a tale of propping his eyes open with match sticks to see the compass. The attacks were worse in the summer when the "weed" was more active, and were often preceded by a headache for a few days. In 1942 he was at home in bed because the rash continued to weep for two months. One wonders why he kept returning to the "weed" which he knew was his enemy but he owned the trawler and had to go for fish. There does not seem to be any evidence of the attacks diminishing; he was better when based at Fleetwood during the war, though it was there that he gave up the struggle and sold his boat in 1953.

When first examined he presented with excoriated papules on the outer surfaces of the forearms and to a less extent on the backs of the hands, with swelling of the arms and legs. No general medical cause was found for the swelling, which pitted only a little on pressure and which had disappeared by the time he was patch-tested a year later. Full haematological and serological examination was normal and of a total of 6900 WBC 4% were eosinophils.

A patch test to a small portion of the "weed" produced irritation from 8 hours onwards and a weeping patch of dermatitis at 48 hours. The arms and face relapsed at 5 days and looked like a case of contact dermatitis with considerable swelling. Bonnevie reported a positive patch test from the "weed" as well as the expressed juice. He mentioned carrying out control tests on normal subjects with negative results. The sea chervil, a seaweed-like Bryozoa colony, is an animal, and Bonnevie pointed out that it is very rare for an animal to be the cause of contact dermatitis.

As would be expected, patients suffering from the disorder are seen mainly at Grimsby and Lowestoft where the fish is landed from the Dogger area. Lanny (1956) states that the condition is seasonal and usually occurs between April and November. He sees his patients at Grimsby and so far all have reported contact to "curly weed" specifically. He mentions that the Danish authorities warn against attempting desensitization of those who are very sensitive, as severe reactions may develop with the smallest dose of allergen.

The patient's skin has cleared considerably in the last three years but he occasionally develops transient patches of constitutional eczema on the fingers, especially in warm weather. X-ray therapy and local application of hydrocortisone have obviously helped him. Since retiring he has not been near the docks at Fleetwood. Before then, even going on to the fish wharves at Grimsby would make his face burn and tingle, and sometimes start an attack. He also thinks that the same applied to Fleetwood but this might have been due to conditioned reflexes. His true degree of sensitivity is shown by his story of waking up one night in his cabin with irritation and swelling of the skin around the eyes and finding out afterwards that he had been crossing the North Dogger.

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THE QUESTION OF URINARY HYPERACIDITY IN SEBORRHOEIC DERMATITIS.

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IN 1918, Barber and Semon published a paper in which they reported a high degree of urinary acidity as one of the primary chemical changes associated with seborrhoeic dermatitis. Since that time there have been no further reviews to expand this observation. Feeling that such an investigation would be of value, we carried out morning urine collections from all new patients attending the Skin Clinic at St. Bartholomew's Hospital in a period from 1 May to 31 August 1956, and tested the pH of each urine. Our results have been summarized in the accompanying table.

Diagnosis.	Number of cases.	Range of pH Values.	Mean pH Value.
Seborrhoeic dermatitis.	50	4.0-8.2	6.0
Acne vulgaris	34	4.3-8.6	6.7
Eczema	92	4.0-9.0	5.5
Skin infections	21	4.0-7.8	5.5
Miscellaneous group	160	4.3-9.0	6.6

From this we may observe that the range of pH values is essentially the same for cases of seborrhoeic dermatitis and acne vulgaris as for a miscellaneous group of dermatoses, including such disorders as neurodermatitis, psoriasis, verruca, carcinoma. The mean pH values found in these cases show an insignificant difference, with the exception of those with eczema or skin infections, in whom the mean pH values of urine are lower.

One would therefore conclude from this group of 357 cases that there is no significant difference in urinary pH levels between those in the seborrhoeic group and the remainder of the patients attending the clinic.

I should like to thank the nursing staff in the Skin Clinic at St. Bartholomew's Hospital for their conscientious assistance in this study.

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PLANTAR WART.

RECURRENCE AFTER CURETTAGE UNDER LOCAL ANAESTHESIA.

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REPORTS from many clinics have drawn attention to the high incidence of warts. The statistics from eleven hospitals in Northern Ireland in which skin clinics are held show that warts of various kinds account for about 17% of the total number of patients attending. In these clinics, as elsewhere, most warts are dealt with by curettage under local anaesthesia, and it is usually only when the patient presents many lesions that arrangements are made for admission to hospital so that they may be removed under general anaesthesia.

Curettage under local anaesthesia when properly performed is generally satisfactory, and the recurrence rate is low, but I have noted a higher incidence of recurrences after this procedure in my own cases where the wart has been situated on the plantar surface of the heel. A recent case suggests a possible explanation for this.

A doctor developed a wart on the plantar surface of the right heel near the posterior edge. As it was his practice to remove warts by curettage under local novocaine anaesthesia in his own patients he attempted to treat his wart himself in this way. He inserted the hyperdermic needle from above and at some distance from the wart, beginning the injection on the back of the heel distal to the attachment of the tendo Achillis and completed the injection into the base of the wart. He then removed the lesion by curettage.

One month later he consulted me with a recurrence of the wart in the original site. I removed it again with xylocaine anaesthesia. I introduced the needle a few millimetres to the side of the wart on the plantar surface and ringed the lesion with a scalpel (but did not include the needle puncture). The wart was then removed intact by curettage. The bleeding points were touched with the electro-cautery and the cavity swabbed with trichloroacetic acid. There was no sign of any additional warty tissue on the heel.

Healing was uneventful, but after 10-14 days, pain recurred on the posterior part of the heel at the site of his original injection. He consulted me again after a further two weeks (i.e. two months after the original injection) and he then had a line of warty tissue in the track of his needle puncture. There was also an early warty point at the site of my injection. The original wart had gone. I admitted him to a nursing home and removed the warty tissue by curettage under general anaesthesia. There has been no recurrence.

This case supports my previous view that recurrences are more liable to take place in plantar warts on the heel where there is a dense mass of tissue to be penetrated by the hyperdermic needle. The track caused by the needle

puncture may allow virus to spread and cause apparent recurrence. Subsequent similar cases support this view.

It is clear, therefore, that when treating warts by curettage under local anaesthesia, the needle should be introduced as near the lesion as possible, and the needle track as well as the warty material should be removed in the subsequent curettage.

This case also shows the likely incubation period for the development of warty change after inoculation.

ROYAL SOCIETY OF MEDICINE.

SECTION OF DERMATOLOGY.

President—Dr. LOUIS FORMAN.

16 March 1956.

Purpuric Lichenoid Dermatitis—Dr. J. S. PEGUM.

Mr. S. W—, aged 64. Occupation : Oil blender.

Nine weeks ago a rash appeared on the ankles. It has gradually spread up to the thighs and buttocks and to the back and shoulders. The rash has been associated with slight aching, burning and a feeling of fullness in the affected parts.

Ten years ago the patient had dermatitis of the legs, which cleared with his doctor's treatment but which left a residual rash on the right ankle. Sixteen months ago he had an attack of retrosternal pain, investigated for coronary disease with inconclusive results.

He drinks tonic water sometimes and takes an occasional aspirin tablet. He was in contact with D.D.T. last summer. His work involves the blending of lubricating oils and additives.

Examination shows scattered and grouped papules 1–2 mm. in diameter, some bright red, on the dorsa of the feet and a scaly red thickened skin on the internal aspects of his ankles. It is a confluent and grouped papular and purpuric eruption, bright red and brown-orange. There is some scaling on the legs. On the thighs there is a purpuric macular and papular rash in a reticular pattern. On the abdomen there are pink oedematous lesions with an occasional papule. The back shows an erythematous and oedematous rash with purpura on the shoulder and posterior axillary folds. There is an erythematous and purpuric eruption on the arms. Blood pressure 190/100.

Investigation.

Blood count (22.ii.56). Hb 94%, 13.9 g. per 100 ml. R.B.C., 4.9 millions. Leucocytes, 4200. Bleeding time, 2 mins. Coagulation time, 2 min 45 secs. Platelets : Plentiful.

Serum proteins (5.iii.56).—Total 7.0 g. per 100 ml. Albumin, 4.7 g. per 100 ml. Globulin, 2.3 g. per 100 ml. Cryoglobulin, *nil*.

Chest x-ray.—*Nil* abnormal detected.

Urine.—Normal.

Hess's test (3.iii.56).—Positive.

Patch tests.—Working overalls : Negative. Long pants : Negative. Sedormid saturated solution in propylene glycol : Negative.

Biopsy.

Biopsy from knee region : The horny layer is for the most part normal, but there is one area of parakeratosis. Parts of the prickle cell layer show spongiosis and liquefaction of the basal cell layer. In the papillary region there is capillary dilatation and red cell extravasation, also an infiltrate of mononuclear cells with occasional giant cells. In the deeper dermis there is perivascular infiltration and proliferation of the blood vessel cells. Iron stain shows a small amount of extra-cellular haemosiderin.

Comment.

I have called this condition purpuric lichenoid dermatitis, following Kierland and Montgomery (1951) who point out that Schamberg's progressive pigmentary disorder, Hutchinson's angioma serpiginosum, and Gougerot and Blum's pigmented purpuric lichenoid dermatitis and, to a lesser extent, Majocchi's purpura annularis telangiectodes are very similar clinically and histologically. If one has to come down from the morphological fence I suppose I should call this pigmented purpuric lichenoid dermatitis of Gougerot and Blum.

Coming to the even more vexed question of aetiology, we have enquired about textile contacts. He has not been in contact with khaki, but the long pants that he wears winter and summer are a mixture of wool and cotton. Sneddon (1952) has reported this condition in a patient who wore a similar mixture. As in Sneddon's patient, patch tests are negative. We have also patch-tested him to Sedormid using Ackroyd's technique (1949). This test was negative. There is no history of taking Sedormid; but Sedormid is a urea derivative and urea-formaldehyde resins are used as textile finishes and we thought there might be cross sensitivity. There is a history of contact with D.D.T. but too long ago to explain his eruption. We are planning to test him with the oils and additives that he comes in contact with at work if we can get his firm to produce some samples—which we have not been able to do yet.

There is one other condition that might give a similar clinical picture. This is purpura hyperglobulinaemia described by Waldenström (1948), a very chronic purpuric condition which in three of his patients had lasted 8, 14 and 16 years respectively. It begins as very small purpuric lesions on the legs and is associated with increase in the globulin fractions in the blood, the so-called macroglobulins or cryoglobulins. My patient's serum proteins are normal and cryoglobulins are not present.

Addendum June 1956.—Patch tests to the 8 oils and additives which the patient blends at work were negative.

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DR. H. R. VICKERS: My colleague Dr. I. B. Sneddon is interested in this condition and I have seen with him a good deal of this sort of case. One feels that it is essentially a toxic capillaritis caused by many different toxins. The localization on the legs appears to be a pure gravitational effect. Only two days ago in Manchester I saw a carbromal eruption similar to this in a man who wears an elastic stocking for varicose veins on one leg and that leg was free from the eruption, as if the pressure of the stocking had prevented the appearance of the eruption. This must be an old disease; it cannot be a new entity although it has been described by different names in the past.

DR. B. C. TATE: I should regard this as in the category of Schamberg's disease. It lasts for varying periods and clears up without any special treatment. It seems to occur in people in all kinds of occupation, clerical as well as manual, and I do not think there was any history of drug-taking in my cases. I do feel that it may be toxic. A girl aged about 20 was shown at this Section by Dr. Goldsmith a good many years ago where the eruption spread right over the trunk. He pointed out that there was a continuously slightly raised temperature suggesting that there might be some toxic factor present.

DR. C. H. WHITTLE: I would like to ask Dr. Vickers if Dr. Sneddon discovered the agent in the textile and if it was washed out in the first washing—I believe that was so. Can Dr. Pegum or Dr. Vickers carry us any further on that point?

DR. J. S. PEGUM: Dr. Sneddon said patch tests with this cotton and wool mixture were negative. He mentioned that after his patient had the condition for 6 months it cleared up. He thought that new clothes are much more likely to be culpable than old ones and he did not find out what it was specifically in the clothes that was responsible.

THE PRESIDENT: There were numerous cases thought to be due to dye or dressing in the material of khaki uniforms. Positive patch tests with this material have been reported,

Dr. J. E. M. WIGLEY: I remember one case in which the offending material was nylon but I could not go further than the fact that the eruption appeared shortly after the stockings were worn and disappeared when they were left off.

Dr. E. J. MOYNAHAN: I have had a patient who was sensitive to the dye and not to nylon. The eruption was very like that in the case which has been shown. The clue to the diagnosis was the disappearance of the rash when she stopped wearing stockings in the summer. Brown lisle stockings and brown suede shoes would also provoke the eruption, so it was obviously the dye rather than the material. A week later a nurse came to out-patients with a purpuric rash confined to the stocking area and when she stopped wearing stockings the rash disappeared.

Dr. G. A. HODGSON: Dr. Hellier and I had a lot of experience with khaki shirts; there were two factors, the dye or mordant factor and the pressure factor. The majority of the eruptions seemed localized by factors like lying in bed, e.g. on the sides or sacrum. We had a patient in Cardiff with a khaki shirt, he left it off and the rash got better. He joined the tramways and got another eruption because the tramways people wore blue shirts which were khaki shirts dyed, so that it was exactly the same thing. It is in the cheaper form of article because the dyeing process is done rapidly, rinsing is not carried out properly and the garment is put on the market with remains of the dye or intermediates in it. It comes out after the first washing as a rule. I do not know what the dye is.

Dr. E. COLIN-JONES: During the war Dr. Twiston Davies published a paper on this subject; he concluded that it was the chrome derivative used as mordant in the dyeing process.

Dr. PETER SMITH: In khaki dermatitis there is no doubt that the distribution of the rash was determined by pressure. There seemed to develop first a dermatitis and then purpura was found in the scratch lines; although the distribution was determined by pressure there was generalized capillary fragility. One could demonstrate it by picking up a fold of the skin and pinching it; this always produced purpura and was a more valuable test than Hess's test. When they came to hospital and wore blue clothes the purpura disappeared but it recurred when they went into khaki again.

Dr. J. S. PEGUM: This condition has also been reported to follow the use of stocking paint where you have the colour without the pressure.

Lupus Erythematosus Arising on the Site of Previously treated Lupus Vulgaris.—Dr. N. A. THORNE.

When I first heard of chronic discoid lupus erythematosus occurring in a patient suffering from lupus vulgaris I felt that this must be just another of those accidental findings that abound in medicine. It was not until I had witnessed the development of chronic discoid lupus erythematosus in two patients whom I had known to have had previous unmistakable lupus vulgaris that I began to feel that there must be some definite connection between the two diseases.

At the London Hospital we have preserved the notes of all patients suffering from lupus vulgaris who have attended the Light Department since its inception in 1903. It would have taken too long to examine all these notes. However, I have searched during the past three years and out of 302 patients who have suffered from lupus vulgaris I have found documentary evidence of subsequent chronic discoid erythematosus in 11. In each lupus erythematosus has appeared in a patient who either had suffered or was still suffering from lupus vulgaris. I was unable to find the reverse phenomenon either in patients at the London Hospital or in any of those reported in the literature. As the notes of some of the cases are incomplete and also because I did not personally witness the transition from one type of lupus to the other I will confine my observations to the two cases already mentioned.

CASE NO. 1.—Mrs. R. E., aged 56, developed lupus vulgaris on the face at the age of 4 years. In spite of 18 years' treatment with acid nitrate of mercury the condition continued to spread. By the age of 33 years there was considerable involvement of the nose, the right side of the face and the lobe of the right ear. Two years later as a result of Finsen light treatment, all the affected areas became inactive. Subsequently there were a number of minor relapses, each responding to further Finsen light treatment.

In January 1953, "apple jelly" nodules and scaliness appeared in the centre of the right cheek. A course of isoniazid 300 mg. daily for 6 months resulted in complete clinical resolution of the lesions. Eighteen months later, central ulceration occurred in the area treated with isoniazid, to be followed by scaliness and erythema. A

biopsy confirmed the presence of chronic lupus erythematosus and did not show any tuberculous giant-cell systems. The patient has begun treatment with chloroquin 400 mg. daily.

CASE No. 2.—Mr. T. McG—, aged 36, first developed lupus vulgaris 11 years ago on the left cheek. “Apple jelly” nodules were present and the acid nitrate of mercury test was positive. In 1950, he was treated with the Finsen-Lomholt lamp and oral calciferol and the lesions completely resolved after 3 months. He resided in Bulawayo from 1951 to 1954 and when next seen in March 1954 there was an area of active lupus vulgaris on the left cheek. He was given oral isoniazid 400 mg. daily for 77 weeks and in addition local injections of calciferol in March and August, 1955. Four months after starting isoniazid the affected area was distinctly flatter but no further resolution occurred and nodules were identifiable on diascopy until February 1955.

In September 1955, the lesion was scaly and erythematous and lupus erythematosus was diagnosed clinically. A biopsy was consistent with this diagnosis and failed to show any tubercles. Chloroquin 400 gm daily was given orally. After 3 weeks' treatment he was greatly improved and at the end of 7 weeks the lupus erythematosus had virtually cleared. He is unable to be present to-day as he has moved from the London area.

Similar cases are reported in the literature, the majority by German authors. Spitzer (1907) described a man aged 37 years who first developed lupus vulgaris on the left cheek which was confirmed histologically and by culture. Later, lesions clinically and histologically typical of lupus erythematosus appeared on the right cheek and elsewhere on the face. He stated that he had only been able to discover two earlier case reports, one by Besnier (1891) and the other in a thesis by Lacavalerie (1895). Zieler (1924) described a case in which lupus erythematosus of the scalp and lupus vulgaris of the face had been present side by side for 20 years.

Leloir noted the variety of lupus vulgaris known as lupus vulgaris erythematoses. I do not feel that either of my two cases fall into this category.

I will not discuss the aetiology of lupus erythematosus. I do, however, feel that its occurrence at a site previously affected by lupus vulgaris suggests that tuberculous infection has some definite aetiological relationship. Possibly a trigger mechanism is necessary to precipitate the onset of the lupus erythematosus. In these two patients no obvious trigger was apparent, but it is interesting to note that each received large doses of ultra-violet light some years previously with none but beneficial results.

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DR. BRIAN RUSSELL: It seems to me that there are four possible explanations for this phenomenon. It could have been lupus erythematosus from the beginning with improvement during treatment with isoniazid; but several experienced dermatologists thought it was typical lupus vulgaris and the colour transparency we have seen today certainly supports this earlier diagnosis. It could be a fortuitous coincidence of lupus vulgaris and lupus erythematosus in the same area. It could be that the two diseases are aetiologically related. Or it could be that treatment of lupus vulgaris with U.V.R. or x-rays can predispose to the development of lupus erythematosus in that position, just as exposure to natural sunlight can predispose to that disease.

I believe this is the true explanation. Recently I saw a woman who told me that she had been treated for scrofuloderma and lupus vulgaris on the face 30 years before with ultra-violet irradiation. The exposure had been excessive and the face was burned and blistered. Subsequently lupus erythematosus developed in this situation and persisted. She had typical chronic discoid lupus erythematosus and it responded well to treatment with chloroquin.

DR. G. B. DOWLING: I think the diagnosis of lupus erythematosus is open to question. This type of red scaly scarred lesion is seen in lupus vulgaris fairly often, in patients who have been treated with x-rays. She said she had been treated with x-rays in 1924,

Dr. SYDNEY THOMSON : I would have thought that it was a definite radio-dermatitis. She is convinced that she was treated with x-rays in her early thirties.

Dr. J. SAVAGE : When I took up my present appointment patients with lupus vulgaris were being looked after by the public health people, and treated with local U.V.R. This treatment had gone on for months and years. When I arrived, they started referring these patients to me, and amongst them were many with lupus erythematosus : although some of them had had local U.V.R. for a considerable time, not one had been aggravated by this treatment, and several patches were healed and pigmented.

Dr. THORNE : I considered that the histology of the affected areas was more in favour of chronic lupus erythematosus than of radiogenic changes. Only in the female patient was there a history of x-ray irradiation. The comparatively speedy clearance of the lesion with chloroquin in the male patient is certainly consistent with a diagnosis of lupus erythematosus. I am unaware that chloroquin has any effect on radiogenic scarring.

Nodular Panniculitis with Atrophy.—Dr. P. A. J. SMITH for Dr. BRIAN RUSSELL.

Mrs. D. M—, aged 37. Housewife.

History.—1944, small lump appeared on the left temple. 1949, similar lump on left wrist : 1950, two lumps came up on outer aspect of right arm ; they were painful but not tender and those on the temple and arm gradually extended. At the same time she had fainting attacks and a productive cough. By 1955 the areas were indurated and depressed and had increased still further in size. In January 1956 hydrocortisone was injected into the erythematous margin three times at weekly intervals. No known injury to the affected sites. No relevant family history.

Past history.—1938, operation for bilateral hallux vagus. Premedication injection given. 1942, attended Royal Free Hospital for blepharitis and retroauricular dermatitis. Radiotherapy given. 1943, bilateral otorrhoea. Tonsillectomy at Royal Free Hospital. Dental treatment at Eastman Clinic. Had four general anaesthetics. 1954, penicillin injections in the thighs.

On examination.—On the left temple and external aspect of right upper arm there are depressed areas at the sites of previous nodules. These were described in 1955 as indurated, and attached to skin but not to muscle. They were originally diagnosed as neurofibromata.

Special investigations.

X-ray chest (1950) : calcification right hilum, otherwise clear. E.S.R. (1955) : 7 mm. in one hour. Skin biopsy from right arm (August 1955) : Predominantly lymphocytes, but also plasma cells, giant cells and fibroblasts in degenerating areas of fat. Histocytic and lymphocytic infiltrate extends into the media of one small artery. Urine : normal.

Comment.

No cause can be found for these areas of panniculitis and fat atrophy. While it is possible that a premedication injection may have precipitated the lesion of the right arm, neither injury nor injection can account for that on the temple. The thighs, where penicillin injections have been given, are normal.

Carotinaemia.—Dr. E. LIPMAN COHEN.

Miss M. W—, aged 37.

History.—Her menses began at the age of 13. Since then she has had a number of episodes of amenorrhoea, one lasting 2½ years and others lasting 4–5 months. At other times there has been oligomenorrhoea, usually with an irregularly increased interval between the periods. Her only other complaint is tiredness.

Since the age of 16 her friends have commented on the yellow colour of her skin. This colour is variable but never disappears completely. It is always most intense on her palms. She is not much concerned about it.

Past history.—Age 12, fractured skull. She has been obese at times, usually when there have been no periods, but keeps her weight under control with diet.

Diet.—She is careful with her diet because of her tendency to obesity. She usually has at least one good portion of carrots daily and plenty of vegetables. She stopped eating carrots for two months and the pigmentation became very faint. She has been asked to eat plenty of carrots for a few days before the meeting.

Examination.—A healthy-looking woman. General examination, normal. B.P. 150/90. Hands, rather large and horny. Yellow pigmentation is most marked diffusely over the palms. Other areas affected are the soles, under the shoulder straps, between and under the breasts, over the hips, and under the chin. The mucous membranes are not involved.

Investigations.

March 1953 : Visual field, normal. Vaginal smear, normal oestrogenic activity. Haemoglobin, 14.5 g./100 c.c. W.B.C., 6600. 17-ketosteroids, 1.9 mg./day. Urinary gonadotrophins, activity present.

December 1953 : X-ray of pituitary fossa, normal. Serum cholesterol, 346 mg./100 ml. The serum cholesterol level came down to normal after treatment with thyroid ; she takes 2 grains daily.

April 1955 : Serum sodium, 330 mg./100 ml. (144 m. eq./litre). Serum potassium, 18 mg./100 ml.

June 1955 : 17-ketosteroids, 8 mg./day.

December 1955 : Serum very yellow. Serum beta-carotene—340 micrograms/100 ml. (normal range up to 200 micrograms).

Urine.—No sugar.

Baelz is said to have described this condition in 1896 and to have called it “*aurantiasis cutis*”. Kaufman (1933) states that Baelz was a German physician living in Japan and that it is not known where his account of the disorder appeared. The name “*carotinemia*” was introduced by Hess and Myers (1919). Hernando (1927) called the clinical condition “*carotinodermia*”. Edwards and Duntley (1939) showed that carotene was normal constituent of the stratum corneum and that it was thus one of the pigments giving rise to the normal colour of the skin. They found that there was much more carotene in the horny layer of women than of men.

Carotinaemia is usually thought of as being very rare but Greene and Blackford (1926) state that it is often seen in diabetics and Bertin, Boulanger, and Huriez (1944) reported a series of 30 cases seen during 1½ years.

Carotene is the precursor of vitamin A. Many fruits and vegetables, especially carrots, oranges, spinach, pumpkins, yellow squash, parsnips and palm oil contain it. Transformation into vitamin A takes place in the liver. A small excess in the serum does not necessarily cause discoloration of the skin. In carotinodermia the palms and soles—the most horny parts—are always the most severely involved. It is commoner in children than in adults.

The vast majority of the patients described have belonged to one of two groups. Alimentary carotinaemia occurs when very large amounts of those fruits or vegetables that contain carotene are eaten for considerable periods of time. It seems that some other factor must be present because, although the condition has been produced experimentally both in man and animals, most vegetarians have skin of normal colour. The other group is those suffering from diabetes mellitus in whom the condition is not rare. Other disorders in which it is said to occur are hypothyroidism, nephritis, and disorders of the liver.

The only symptoms of which these patients complain are discoloration of the skin and tiredness.

This patient has alimentary carotinodermia, the form usually found in children. She has always eaten large quantities of carrots and the yellow colour of the skin

almost disappeared when she stopped eating them. She has no sign of diabetes but her hypothyroidism probably plays a part.

I wish to thank Dr. R. M. B. MacKenna for permission to present this patient.

REFERENCES.

- BERTIN, E., BOULANGER, P. and HURIEZ, C. (1944) *Pr. méd.*, **52**, 2.
 EDWARDS, E. A. and DUNTLEY, S. Q. (1939) *Amer. J. Anat.*, **65**, 1.
 GREENE, C. H. and BLACKFORD, L. M. (1926) *Med. Clin. N. Amer.*, **10**, 733.
 HERNANDO, T. (1927) cit. KAUFMAN, E. (1933) *vide infra*.
 HESS, A. F. and MYERS, V. C. (1919) *J. Amer. med. Ass.*, **73**, 1743.
 KAUFMAN, E. (1933) *Jadassohn's Handbuch der Haut-und-Geschlechtskrankheiten*, **4/2**, 1090. Berlin : Springer.

Gp. Capt. H. E. BELLINGER : Dr. Lipman Cohen asked how common was this condition and I am sure a good many members of the Section who were in the Royal Air Force during the war may remember the incident that occurred in Blackpool in 1942 when several hundred airmen were found to have carotinaemia. Everyone thought a jaundice epidemic had broken out until it was realized that the vegetable portion of their diet was 80% carrots, the cheapest vegetable at the time. The Blackpool landladies were feeding the airmen with carrots all the winter, and when the serum was separated from the blood cells it was like the heart of a carrot to look at.

The other interesting thing associated with this is that towards the end of the war the German Air Force used extracts of marigold petals to improve the night vision of their night fighters and they found that it produced carotinaemia in their pilots. This was revealed in some secret documents I perused after the war.

Dr. BRIAN RUSSELL : During the war I saw a greengrocer's wife with carotinaemia. She used to sit outside the shop to prevent larceny of the vegetables that were exposed for sale. She nibbled raw carrots all day and was a much brighter yellow than Dr. Lipman Cohen's patient.

Should not Dr. Lipman Cohen also have mentioned eunuchoidism ? There is said to be a failure to convert carotene into vitamin A in eunuchoidism, nephrosis and myxoedema.

Dr. E. J. MOYNAHAN : I disagree. The pigment of eunuchoidism is said to be melanoid. In the paper by Edwards and Duntley the five pigments of the skin are discussed and it is said that the eunuch is deficient in melanin. I have seen eunuchs develop suntan after being given testosterone in the winter. The colour of the skin of eunuchs is peculiar, it is not like carotene. I have seen patients who dieted themselves for some reason and drank tomato juice *ad lib.* who developed an even more canary colour than the patient presented to-day. They do not often attend out-patients : they come with something else and one notices the colour and finds that they are slimming or dieting for some other reasons. Vegetarians are prone to this.

Dr. H. HABER : There is another condition which brings about yellowness of the palms and that is pityriasis rubra pilaris. It has been found that it is due to the presence of carotene in the horny layer.

Acanthosis Nigricans Juvenilis.—Dr. C. D. CALNAN.

F. M.—, man aged 25.

History.—Since the age of one year, this man has noticed discoloration of his skin over most of his body, together with thickening on the palms.

Examination.—Almost all areas of his skin show sheets of warty thickening with black pigmentation. The flexures of the groins, axillae, and the neck are most affected. On the arms and some other areas there is a tendency to a linear pattern. There is thick hyperkeratosis of the palms, but little on the soles. The horn shows an increase in the lines of the skin and some fissures.

On the backs of the hands and forearms are lesions like flat warts or seborrheic keratoses. On the little finger of one hand the wart appears to be continuous with the hyperkeratosis of the palm.

Mouth and mucous membranes are normal.

General examination of the heart, lungs, and abdomen is negative. No other physical abnormality detected.

Histology.

Section 1.—The epidermis is thrown into folds. This is due to a hyperkeratosis which not only involves follicles but is also seen to dip deeply into the epidermis extrafollicularly. The rete pegs show finger-like elongations leading, therefore, to

elongation of the papillae. The whole structure in this way attains a very characteristic appearance. The basal cell layer is hyperpigmented. Within the upper corium a mild round-cell infiltration is demonstrable.

Section 2.—The epidermis shows very marked hyperkeratosis, granulosis and acanthosis with elongated and fused rete pegs. The papillary layer shows dilated vessels and a mild infiltrate consisting chiefly of fibroblasts and a few round cells. The histological appearance is that of acanthosis nigricans.

Comment.

This man is an example of widespread acanthosis nigricans of the benign type, beginning at birth or in infancy. He shows the isolated warty lesions on the backs of the hands and in one place such a lesion is continuous with thick hyperkeratosis of the palms. There is no morphological difference to distinguish the benign and malignant types except that mucosal involvement is almost unknown in the benign form.

This man has no clinical evidence of malignant disease; no special examinations have been made. Nor has he evidence of other congenital abnormality, or endocrine disorder such as pituitary basophilism as is sometimes associated with this condition.

Dr. S. C. GOLD: Occasionally these cases follow upon a head injury, presumably resulting in damage to the pituitary fossa.

The PRESIDENT: Most of the cases appear at puberty.

Dr. C. D. CALNAN: Yes, but it is recognized that they may start at birth, and increase at puberty. All sorts of congenital and acquired associated abnormalities have been described. The skull injury may possibly have damaged the pituitary; quite a few cases have been shown to be associated with pituitary basophilism. Such a case with post-mortem findings was reported by Rothman (1954). The thesis was put forward that the juvenile cases may be caused by some pituitary disorder; many of the patients have no other abnormality, as my patient.

REFERENCE

ROTHMAN, S. (1954) *J. Amer. med. Ass.*, **156**, 242.

Dr. E. J. MOYNAHAN: I think Helen Curth is the leading authority on acanthosis nigricans and she would say that almost without exception cases before puberty were benign, and if one thinks about it, one realizes that all the cases which appear later are associated with malignancy, usually in the intestinal tract. Helen Curth would look upon the former variety as a genetic abnormality on its own, a recessive trait that crops up from time to time and is not related to any other abnormality, but we need more pedigrees before we can be sure that there are two distinct entities.

Polycythaemia with Gangrene of Fingers.—Dr. CLARA M. WARREN.

Mrs. L. B—, aged 65.

20.ii.56.—She was admitted under the care of Dr. Hugh Gordon for investigation and treatment of severe "chilblains" and pain in two fingers of the right hand.

History of chilblains since childhood; always feels cold in winter; never comfortable in the heat of summer.

Family history.—Her father and two brothers are stated to have had similar colouring and tendency to chilblains. Her father died at 77 and her mother at 82, each from a stroke. Her son was killed at 24. He had been healthy.

Appearance.—Colour of face good, but congestion of malar areas, ears, nose and lips. Tips of index and middle fingers of right hand show dry gangrene which followed blister formation. The skin is warm but congested in colour. Raynaud's phenomenon is present in the middle finger.

Blood examination.—Haemoglobin 135%, 19.9 g.%. Red cells 7,000,000 per c. mm. (polychromasia) C. I. .9. White blood cells, 13,200 per c. mm., myelocytes 3%, metamyelocytes 3%, polymorphs 85%, lymphocytes 9%. Platelet count 505,000 per c. mm.

Spleen and liver just palpable. B.P. 180/80

Chest has increased lung markings in skiagram.

With rest in bed the gangrenous areas have dried up.

Dr. HUGH GORDON : This patient is being referred for treatment with radioactive phosphorus since it appears that this is the treatment of choice for the condition, and indeed, one of the most satisfactory conditions to treat with radioactive phosphorus. At the Royal Marsden Hospital a series has now been treated numbering approximately 70.

The results of treatment have, on the whole, been very good. Some were entirely controlled and the blood picture restored to normal after one treatment. In most cases, however, further treatments were necessary. The longest period of attendance completely free from symptoms is 4 years after treatment.

Myelosclerosis had supervened in three patients and leukaemia also in two.

Dr. G. B. DOWLING : What is the mechanism of this process ?

Dr. GORDON : I understand that it is generally held that the large increase in viscosity of the blood is responsible. There was, I think, no doubt that cold was a factor here since only one hand was affected, and this was the hand chiefly used in a very cold spell.

The following cases have been published in *Proc. R. Soc. Med.*, **49**, 822.

Juvenile Dermatomyositis.—Dr. C. M. RIDLEY for Dr. BRIAN RUSSELL.

Skin Reactions at Site of Green and Red Tattoo Marks.—Dr. J. A. BONNELL for Dr. BRIAN RUSSELL.

Hydroa Gravidarum.—Dr. HAROLD WILSON.

A short paper on **Rhinoplasty and Diseases of the Skin** was read by Mr. CHARLES HEANLEY. The following cases were shown to illustrate Mr. Heanley's paper :

Rhinoplasty for Carcinoma on Lupus Vulgaris. Rhinoplasty for Sarcoma on Lupus Vulgaris.—Mr. CHARLES HEANLEY and Dr. BRIAN RUSSELL.

Rhinoplasty for Old Gumma of Nose and Palate.—Mr. CHARLES HEANLEY and Dr. AMBROSE KING.

The following cases also were shown.

Connective Tissue Naevus.—Dr. C. M. RIDLEY for Dr. BRIAN RUSSELL.

Hypertrichosis Lanuginosa(Two Cases).—Dr. J. E. M. WIGLEY.

Necrobiosis Lipoidica Diabeticorum Improving under Treatment with Local Hydrocortisone Injections.—Dr. P. J. HARE.

SOUTH WEST OF ENGLAND AND WALES SOCIETY OF DERMATOLOGY

Bristol, 6 October 1956.

Pyogenic Granuloma with Local Recurrences (2 cases).—Dr. C. D. EVANS and Dr. R. P. WARIN.

Case 1.—This schoolboy, aged 10 years, was first seen in February 1954 with a typical pyogenic granuloma over the lower end of the left scapula. This was excised and within a month numerous red raised pin-head lesions had appeared at the edges of the scar and extended from it for 2 inches. A further biopsy was performed. The boy was then shown at the Royal Society of Medicine (Evans, 1954).

Histology.—Both the initial and the secondary lesion showed identical appearances typical of pyogenic granuloma.

Progress.—For 6 months after the secondary lesions appeared they increased in size but no new ones were seen. Since then they have slowly regressed spontaneously and the skin in the area is now normal (Evans, C. D. (1954) *Proc. R. Soc. Med.*, **47**, 1063).

Case 2.—This boy, aged 8, was first seen in May 1956 when he had a typical pyogenic granuloma 1 cm. in diameter on the left shoulder, which had been present for 5 months. This was curetted off but when seen 6 weeks later there were numerous pin-head sized raised red areas in the area around the scar. These have remained unchanged in the 5 months up to the present time.

Histology.—The appearances are those commonly seen in so-called pyogenic granuloma.

Comment.—Both these cases appear to be clinically identical with that described by Sims *et al.* (1948) under the title of Haemangiopericytoma. The histological picture they describe does not appear fundamentally different from our cases. On the other hand the cases described under the title of Haemangiopericytoma by Cole *et al.* (1955) seem to be quite different.

Dr. R. E. BOWERS: If in the common granuloma telangiectaticum (pyogenic granuloma) the stimulus to capillary growth is related to capillary pressure, it is possible that in these cases the pressure change is transferred to the surrounding area of skin after the primary lesion has been removed.

REFERENCES.

- SIMS, C. F., KIRSCH, N. and MACDONALD, R. G. (1948) *Arch. Derm. Syph., Chicago*, **58**, 194.
COLE, H. N., REAGAN, J. W. and LUND, H. Z. (1955) *Ibid.*, **72**, 328.

Stevens-Johnson Syndrome with Intestinal Symptoms.—Dr. C. D. EVANS.

This schoolboy, aged 16 years, had his first attack in 1952. Typical blood crusts were present on the lips; the mouth and tongue were severely ulcerated and very painful. He had conjunctivitis, nasal discharge and urethritis with a meatal ulcer. The only skin lesions were 3 small erythematous macules, surmounted by vesicles, on the chest and 2 on the left thigh. On the fourteenth day of the illness he had abdominal pain and diarrhoea and passed fresh blood and mucus; this lasted for 48 hours. He had had no aureomycin for 4 days, nor was he receiving any drugs. No bacterial cause was found for the intestinal symptoms and agglutination tests for typhoid, paratyphoid and salmonella were negative. The temperature fell on the eighth day, 24 hours after starting aureomycin therapy; sulphatriad and penicillin had been used before this without benefit.

In 1955 he had a second attack, not quite so severe as the first. There were no skin lesions and no abdominal symptoms. Aureomycin was given at the onset but had no beneficial effect. The temperature again fell on the eighth day.

In September 1956, two weeks after extraction of a right upper molar tooth, he developed his third attack. Again there were no skin manifestations. He had abdominal pain and diarrhoea lasting for 48 hours as in the first attack, but no blood or mucus was passed. The severity of the symptoms was slightly less severe than in the former attacks. He received no treatment except mouthwashes and troch. benzocain co. to suck. The temperature again fell on the eighth day.

Investigations.—Chest skiagrams in each attack were normal.

Blood: Hb normal, film normal. No leucocytosis.

Bacteriology of the mouth lesions showed normal flora and no monilia. Urine and stools: normal.

Comment.—In each attack the duration of the pyrexia was 8 days irrespective of treatment. There were minimal skin lesions in the first attack and none in the second and third.

No sulphonamides had been given prior to any of the attacks.

Fresh blood and mucus was passed on the fourteenth day of the first attack and abdominal pain and diarrhoea occurred in the third attack. This has been reported previously by Dresner (1949) and is presumably due to erythema multiforme affecting the bowel.

REFERENCE.

DRESNER, E. (1949) *Lancet*, ii, 1036.

Urticaria Pigmentosa Persisting into Adult Life.—Dr. R. P. WARIN.

This patient, aged 37, started with a brown mark on one foot when 3 months old. New lesions quickly developed and the condition then remained unchanged up to the present.

Examination.—Typical widespread urticaria pigmentosa lesions readily urticating on trauma.

Comment.—The patient was shown as it seems generally accepted that in most, if not all, juvenile cases of urticaria pigmentosa the lesions disappear before puberty.

Blood Dyscrazia during Dapsone Therapy for Dermatitis Herpetiformis.—Dr. L. G. TULLOCH (for Dr. R. P. WARIN).

This girl, aged 3, was first seen in April 1956, when a history was given of blistered lesions around the vulva and later the umbilicus, right axilla and back of the neck. The appearance was that of dermatitis herpetiformis and she was put on sulphapyridine 0.25 g. twice daily. This provided only partial control so she was given dapsone 25 mg. daily and later 50 mg. daily, on which the eruption remained clear for 2½ months. While on this treatment in July, 1956 she developed dyspnoea and cyanosis. She quickly recovered on stopping the drug, but 8 days later the total neutrophil polymorphs were only 975 per c.mm. and platelets were scanty. 10 days later the blood picture was becoming normal.

Comment.—No blood examination was possible at the onset of the attack but the symptoms suggest the possibility of methaemoglobinæmia. There were no symptoms of the granulocytopenia.

Subcorneal Pustular Dermatitis (Sneddon and Wilkinson). Cortisone Therapy Complicated by Severe Acne-like Eruption.—Dr. L. G. TULLOCH (for Dr. R. P. WARIN).

Four years ago this man, aged 76, developed an eruption on his legs similar to lichen planus hypertrophicus. Two years later this cleared but was followed by an

eruption with a slowly advancing pustular margin, with occasional transient vesicles. Later these appeared on the trunk, anogenital area, arms, fingers, scalp and mouth. Some of the patches cleared after a few weeks but new ones developed on the same site or elsewhere. The appearance brought to mind that described by Sneddon and Wilkinson (1956) and the case demonstrated by Sneddon. However, in our case dapsone 50 mg. *t.i.d.* had no effect. The eruption was finally controlled on large doses of cortisone up to 250 mg. per day. On this dose he developed a severe acne-like eruption on the face, and on the back and front of the chest. Comedones and pustules were present.

Bacteriological and mycological studies of the pustular margins showed various common bacteria, almost certainly secondary invaders.

On one occasion there was a blood eosinophilia of 10% but at other times the blood picture was normal.

REFERENCE.

SNEDDON, I. B. and WILKINSON D. S. (1956) *Brit. J. Derm.*, **68**, 386.

BOOK REVIEWS.

PSYCHIATRY AND ALLERGY.*

THIS book deals with psychosomatic allergy as manifested mainly by asthma, "dermatitis", hay-fever, and to a less extent urticaria. No doubt many of the cases referred to are of what dermatologists often call atopy. "Immunologic allergy" as evidenced by positive skin reactions may or may not be present in those who have allergic symptoms: these authors imply that the psychiatric factor is the more essential one, and that this is usually based on maternal rejection and its complex psychological consequences.

Physical treatment by desensitization, sedation, etc. is briefly considered but the greater part of the book is devoted to detailed descriptions of psychiatric mechanisms and to the care and handling of patients and relatives. The style is chatty, with much reference to illustrative cases and dialogue. To the dermatologist it is interesting to see psychiatry with the lid off, but not many of us would feel competent to adopt the authors' methods ourselves. Although the authors are evidently most expert and wise as well as kindly, they say little about the results of treatment and may not therefore particularly encourage the dermatologist to seek help from the psychiatrist in this type of case. But it is an interesting book. F. R. B.

DERMABRASION.†

THE choice of title for this work on surgical planing of the skin is a little unfortunate, the wire brush now being somewhat outmoded and replaced by specially designed cylindrical burrs.

The author points out that Rossignol in 1881 used scarification of the skin in the treatment of acne, rhinophyma, and naevus flammeus. The removal of redundant tissue in rhinophyma is the classical example of "scarless surgery", with epithelial regeneration taking place from the depths of the follicles. Kromayer described dermal planing in 1905 and experimented extensively with cylindrical knives, augers, borers, and burrs. He observed that abraded wounds healed without visible scarring, provided the abrasion was confined to the pars papillaris and did not involve the reticular cutis. Interest in dermabrasion has been revived with the introduction of suitable refrigerants, more rapid motors, and the greater safety provided by antibiotics. Dichlorotetrafluorethane (Freon 114) rapidly freezes the skin without the necessity of using a blower to hasten evaporation. It is said to be non-toxic and, unlike ethyl chloride, it is non-inflammable and non-explosive. Kurtin (1953) improved the methods of refrigeration and introduced the rotating wire brush in place of a sandpapering technique.

The author deals with all aspects of dermal planing, the equipment, the details of technique, the histopathology of healing, anaesthesia, post-operative care of the "planee", complications, indications and contra-indications.

He avers that planing can be continued to the depth where minute herniating buds of yellow fat appear; below this depth, scarring results. The commonest complica-

* *The Practice of Psychosomatic Medicine as Illustrated in Allergy.* HYMAN MILLER and DOROTHY W. BARUCH (1956). London: The Blakiston Division, McGraw-Hill Book Co., Inc. Pp. 196. Price 37s. 6d.

† *Wire Brush Surgery.* JAMES W. BURKS. (1956) Springfield, U.S.A.: Charles C. Thomas. Pp. 154. 68 Figs.

tions are milia, either early and superficial or late and deep. He has not observed keloid formation. Indications include acne scars and active but non-infected acne. In severe cystic acne the objective of the first planing is "marsupialization", while subsequent planings provide cosmetic improvement. Acne rosacea is suitable for treatment, as are also hydradenitis, rhinophyma (12 weeks after removal of the gross excrescences with the cautery knife), traumatic scars and pox scars, but not burn scars. Tattoo marks give comparatively poor results on the forearms, upon which unfortunately much of this form of adornment is carried out. Thin skin is more likely to scar, and skin defective in pilosebaceous and sweat appendages is likely to be slow in healing and may fail to heal at all. The author has also used surgical planing in the treatment of chloasma, freckles and superficial naevi, naevus flammeus, keloids, lupus erythematosus, superficial epitheliomas, "crow's feet", keratoses, adenoma sebaceum, and Fox-Fordyce disease. In xanthelasma, other methods of treatment are preferable.

Contraindications include radiodermatitis, burns, and pyoderma. Psychotic, severe psychoneurotic, and alcoholic patients are not acceptable, and criminals seeking removal of identifying marks must also be rejected.

The book ends with references from the Papyrus Ebers to the present day. It is well worth close study by anyone who wishes to learn the technique of dermal planing.

B. F. R.

ANGIOMATA.*

THIS monograph has been well written by a radiotherapist primarily for her colleagues and students of radiotherapy but the subject very much concerns the dermatologist. Other methods of treating vascular naevi, such as freezing and the injection of sclerosing agents, are briefly described but the main work is concerned with the use of radium and x-rays.

Considerable prominence is given to the dangers of excessive radiotherapy in the treatment of these angiomas and it is, therefore, surprising to find no acknowledgment of the fact that the huge majority of these lesions resolve spontaneously and therefore need not be treated at all. This particularly applies to angiomas of the scalp, those in the pelvic area, and those overlying the breasts, but the author describes the treatment of lesions in all these sites although it is agreed that the dosage suggested carries little risk.

The general recommendation is that doses of 100–150 r should be given once or twice a week to a total of 700–900 r at 120–150 Kv., filtration 2 mm. Al., F.S.D. not exceeding 30 cm. and the surrounding healthy tissue should be properly screened.

Many excellent clinical photographs support the author's practice, and an account of her very extensive experience is a welcome addition to the literature upon this subject.

R. T. B.

TROPHIC DISORDERS OF THE LOWER LIMBS OF VENOUS ORIGIN.†

THE increasing interest taken in vascular disorders of the lower limb is widespread, and to follow developments in this subject familiarity with an ever accumulating body of literature is required. Even the serious reader is almost driven to an easy parochialism. Hence the publication of a book of this type is most welcome. This

* *Le Traitement des Angiomes Chez les Enfants*. SIMONE LABORDE (1956). Pp. 174. 71 Figs. Paris: Masson et Cie. Price 1850 Francs.

† *Les Troubles Trophiques des Membres Inférieurs d'Origine Veineuse*. Edited by JACQUES CHARPY and MARIUS AUDIER (1956). Paris: Masson et Cie. Pp. 375. Figs. 176. Price 2400 Francs.

monograph is a collection of addresses and short papers read at a joint meeting of *La Filiale Marseillaise, la Société française de Dermatologie et de Syphiligraphie* and the third *Journées internationales de Phlebologie* held in 1955. The first contribution (by Jausion, Roussel, Vieville, Sylvestre and Chevrier) is a paper dealing fully with disorders of veins and small vessels and their results. Pigmentary and purpuric changes are well described and the effects of capillary lesions discussed in detail. There is a good bibliography and the illustrations are excellent.

Papers follow on the surgical treatment of varicose veins and of post-phlebitic complications. The results of surgery on conventional lines appear impressive but full information in regard to the follow-up of patients is not given. No mention is made of Cockett's important work on the subject.

There is a long paper on dermatological practice in relation to these conditions. In discussing symptomatic treatment, carried out during "*la phase dermatologique*", complete rest in bed is insisted on and various methods of relieving pain described. Local treatment with antibiotics is advised for ulcers and later elastic bandages may be used. In the next phase of treatment varicosities are injected or ligated or other surgical methods applied. Grafting, Filatov's tissue implantation, massage, and spa treatment are mentioned. The illustrations in this paper are not of high quality.

The remainder of the book consists of about 40 short papers on aetiology, histology, phlebography and treatment, with ulceration as the main consideration. Factors such as arterial disease, capillary changes and obesity are discussed. Many of these papers are useful but it is unfortunate that, with few exceptions, no list of references to authors quoted in the text is given. Nevertheless, this is a valuable book for those who wish to learn the views of French and other continental authors on this subject. An index would have made it even more helpful.

S. T. A.

CURRENT LITERATURE

FACTORS INFLUENCING SKIN REACTIONS IN GUINEA-PIGS. A. NILZEN and K. WILKSTROM. (1955) *Acta dermat.-venereol., Stockh.*, **35**, 416.

The skin reactions of guinea-pigs sensitized to 2:4 dinitrochlorobenzene were examined during exposure to cold and also during administration of cortisone and A.C.T.H. The figures given suggested that there was slight delay in the development of the standard epidermal reaction after exposure to cold.

O. L. S. S.

EXPERIMENTAL STUDIES ON HYPERSENSITIVITY. E. SKOG. (1955) *Acta dermat.-venereol., Stockh.*, **35**, 401.

Detailed results of careful experiments which support the findings of Haxthausen and of Sulzberger who were unable to produce passive transfer of hypersensitivity of skin with dinitrochlorobenzene. The passive transfer of tuberculin sensitivity (which lasted less than 5 days) was obtained with cells from lymph, thymus, spleen, and peritoneal exudates. Methods of measuring the minimum number of cells required to produce sensitivity are described, and the author considers the numbers to be comparatively large.

O. L. S. S.

PATCH-TEST STUDIES. A. I. B. FERNSTROM. (1955) *Acta dermat.-venereol., Stockh.*, **35**, 420.

The author describes in detail his "pressure-patch-test" method which makes use of sponge rubber over the occlusive cellophane or rubber. He claims that this is speedier than other methods, and that the patches are less likely to slip.

O. L. S. S.

DISCOID AND DISSEMINATED LUPUS ERYTHEMATOSUS. A. LODIN and N. THYRESSON. (1955) *Acta dermat.-venereol., Stockh.*, **35**, 429.

This is a follow-up of 512 cases of lupus erythematosus with a good statistical survey. A high incidence of raised E.S.R. was found in both discoid and systemic forms of the disease. The serum gamma globulin was raised in a number of patients with the discoid form.

O. L. S. S.

PSEUDO-XANTHOMA ELASTICUM AND VASCULAR DISTURBANCES. B. BAFVERSTEDT and F. LUND. (1955) *Acta dermat.-venereol., Stockh.*, **35**, 438.

An interesting short review with particular reference to a nine-year-old boy with skin changes who seemed otherwise normal. Oscillography, plethysmography, and arteriography suggested early degenerative changes in the vessels.

O. L. S. S.

MEPACRINE IN ROSACEA. P. INMAN and B. GORDON. (1955) *Acta dermat.-venereol., Stockh.*, **35**, 448.

Good results were obtained in 300 patients, but the authors favour a combination with x-rays and sulphur and salicylic acid ointment.

O. L. S. S.

VITILIGO TREATED WITH CHLOROQUIN. J. CHRISTIANSEN. (1955) *Acta dermat.-venereol., Stockh.*, **35**, 454.

A man with extensive vitiligo obtained relief from his light sensitization when he took chloroquin. This was accompanied by extensive repigmentation.

O. L. S. S.

THE RASH OF RHEUMATOID ARTHRITIS AND STILL'S DISEASE. I. C. ISDALE and E. G. C. BYWATERS. (1956) *Quart. J. Med.*, (N.S.), **25**, 377.

A rash that appears to be specific for this disease occurred in 46 of 166 children with Still's disease and in 7 adults out of over 500 with rheumatoid arthritis. This rash most commonly affects the limbs (97%), trunk (82%) and face (59%), is transient, appears towards evenings and with high temperatures, and does not spread. It may precede other manifestations of the disease by as much as three years.

The characteristic rash is erythematous macules 3 mm. or less in diameter with a slightly irregular margin. Larger lesions with central pallor may occur. They may be slightly raised. They do not itch. The incidence of fever, of acromegaly and lymphadenopathy in children who had a rash is significantly higher than in those who had no rash.

Cortisone, corticotropin, salicylates and antihistamine drugs have no specific effect on the rash.

This rash may be of considerable diagnostic value (1) in cases in which arthritis is slight and (2) when the clinical picture otherwise resembles rheumatic fever.

J. M. B.

THE INFLUENCE OF VITAMIN D₂ ON SOME BODY FUNCTIONS. L. I. GOKINAEVA. (1956) *Vestnik Ven. i Derm. (Moscow)*, 4, 19.

The author is impressed by the hormonal effects of vitamin D₂ (calciferol) given to tuberculous skin patients. A few of 18 patients with amenorrhoea were married women of whom none had been pregnant for 10 years: 6 had a healthy child following treatment. Another patient aged 51 with amenorrhoea for 27 years began after treatment to menstruate again and continued regularly for over 3 years. Other 3 climacteric patients with amenorrhoea for 6 months or longer began menstruating again after 2-2½ months' treatment.

Acrocyanosis was found to improve under the treatment.

No mention is made of the use of isoniazid in lupus vulgaris.

M. S. S.

PUZZLING PERSISTENT PENILE PLAQUES. MARION B. SULZBERGER, VICTOR H. WITTEN and JOHN A. HUNT. (1956) *A.M.A. Arch. Derm. Syph., Chicago*, 73, 101.

This article is devoted to four conditions that may give rise to difficulty in diagnosis; these are, lichen planus, psoriasis, distinctive exudative discoid and lichenoid chronic dermatosis of Sulzberger and Garbe and erythroplasia of Queyrat. The latter two are considered in detail and the importance of differential diagnosis is stressed as the one is benign in course and the latter is precancerous or cancerous.

J. K.

THE VATER-PACINIAN CORPUSCLE IN THE SKIN OF THE HUMAN FINGERTIP. RICHARD K. WINKELMANN and LAMAR S. OSMENT. (1956) *A.M.A. Arch. Derm. Syph., Chicago*, 73, 116.

The observations are confined to the shape and size of the corpuscle and the numbers found in this area of skin. The shape was ascertained by wax reconstructions from serial sections and showed that the corpuscle is much more irregular in appearance than was realised. There were 48 per square centimetre of skin surface and 120 per cubic centimetre of fingertip tissue.

J. K.

ECCRINE SPIRADENOMA. DAVID W. KERSTING and ELSON B. HELWIG. (1956) *A.M.A. Arch. Derm. Syph., Chicago*, 73, 199.

The authors base their study on 134 tumours most of which occurred on the ventral surface and "above the waist". Almost all patients showed a single tumour and the most common characteristic was pain or tenderness. The size of the tumour varied from 0.3 to 5 cm., the colour also varied but a blueish appearance was common. There was no recurrence after complete extirpation and no evidence of malignancy. The microscopic appearances are dealt with in detail and are those of a tumour of the eccrine gland, quite different from the papillary hidradenomas, syringomas, myoepitheliomas and cylindromas.

J. K.

HISTOPATHOLOGY OF CUTANEOUS LESIONS IN SYSTEMIC LUPUS ERYTHEMATOSUS.

MICHEL PRUNIERAS and HAMILTON MONTGOMERY. (1956) *A.M.A. Arch. Derm. Syph., Chicago*, 74, 177.

This article deals with the newer staining methods for studying the skin in lupus erythematosus. Although only systemic lupus erythematosus is dealt with, it is pointed out that the histology of systemic and discoid lesions is similar provided they are four weeks old. There are no striking conclusions but the article is useful for reference for the various methods employed.

J. K.

SENSITIZATION DERMATITIS DUE TO CARROTS. JOSEPH V. KLAUDER and JOHN M. KIMMICH. (1956) *A.M.A. Arch. Derm. Syph., Chicago*, 74, 149.

A series of 13 cases of dermatitis from handling carrots is reported. All showed positive patch tests to raw carrot but of two tested with boiled carrot, one showed a positive reaction and one a negative. Cross sensitization to other botanically related vegetables such as parsnips and celeriac was noted in some cases. Phytophotodermatitis is discussed, but it does not appear that, though other related umbelliferae are known to be a cause, carrots have been shown to

cause this condition. It is suggested that the edible umbelliferae should be considered as a cause of dermatitis of the hands in housewives or even of cheilitis and perioral dermatitis.

J. K.

REACTIONS TO CHLORAL HYDRATE. HERBERT B. CHRISTIANSON and HAROLD O. PERRY. (1956) *A.M.A. Arch. Derm. Syph., Chicago*, **74**, 232.

Reactions to chloral hydrate are admittedly rare, but the authors on the strength of 7 cases of their own and a survey of the literature, have classified them under 10 headings and several sub-headings giving a total of 22 different types of rash this drug may cause. In addition delirium and somnambulism may occur.

J. K.

PIGMENTATION IN THE BLOCH-SULZBERGER SYNDROME (INCONTINENTIA PIGMENTI). LOUIS RUBIN and S. WILLIAM BECKER, jr. (1956) *A.M.A. Arch. Derm. Syph., Chicago*, **74**, 263.

The pigmentation seen in this syndrome is not post-inflammatory but is a separate symptom. The dermal chromatophores are the result of normal pigment formation and this histological picture may be seen in other diseases such as photosensitization reactions, fixed drug eruptions and Addison's disease. No explanation is given of the bizarre patterns seen in incontinentia pigmenti.

J. K.

HISTOGENESIS OF BASAL-CELL EPITHELIOMA. OTHELLO D. SMITH and MARTIN A. SWERDLOW. (1956) *A.M.A. Arch. Derm. Syph., Chicago*, **74**, 286.

The authors have reviewed 147 biopsies from basal-cell tumours in 122 patients and found "keratin-cell complexes" in 144 and suggestive pearl formation in the other three. They consider that this supports the view that these tumours arise from "either epidermal germ buds or hair anlage".

J. K.

A SERUM ANTICOAGULANT FACTOR IN SYSTEMIC LUPUS ERYTHEMATOSUS. SHELDON SWIFT. (1956) *A.M.A. Arch. Derm. Syph., Chicago*, **74**, 296.

An anticoagulant factor may be present in the serum of patients with systemic lupus erythematosus even with no history of excessive bleeding. It is sufficiently powerful to inhibit coagulation even in a dilution as low as 1 in 10. It seems to inhibit thromboplastin and is located in the serum gamma globulin. In one patient this factor disappeared during a remission, an extremely unusual occurrence with anticoagulants of this type.

J. K.

AMODIAQUIN (CAMOQUIN) IN THE TREATMENT OF CHRONIC DISCOID LUPUS ERYTHEMATOSUS. ROBERTS B. PAPPENFORT and JAMES H. LOCKWOOD. (1956) *A.M.A. Arch. Derm. Syph., Chicago*, **74**, 384.

This, the latest of the antimalarial drugs to be reported on, showed good results in 9 patients resistant to mepacrine and chloroquin; side effects were slight. The dose used was 0.2 g. twice daily for a week, then once daily.

J. K.

MEPROBAMATE (MILTOWN) AS ADJUNCT IN TREATMENT OF ANOGENITAL PRURITUS. OSCAR J. SOKOLOFF. (1956) *A.M.A. Arch. Derm. Syph., Chicago*, **74**, 393.

A series of 29 patients with idiopathic pruritus ani or vulvae was divided into three groups. All were treated with local hydrocortisone and x-rays but in addition one group was given phenobarbitone and one meprobamate; the third group was used as a control. While phenobarbitone controlled the itching patients suffered from "dopiness," meprobamate helped to "bring the patient's anxieties and tensions under control more rapidly than would be otherwise possible" without side-effects. Of the series of 10 controls, 7 were controlled by x-rays and hydrocortisone and 3 uncontrolled till meprobamate was given in addition.

J. K.

CONCERNING THE IDENTITY OF HYDROA VACCINIFORME AND CUTANEOUS PORPHYRIA. M. BOLGERT, J. CANIVET and J. LÉPINE. (1956) *Ann. Derm. Syph., Paris*, **83**, 18.

The authors point out the clinical differences between these two conditions, the former affects covered areas and the buccal mucosa, showing umbilicated vesicles and leaving indelible scars. They believe hydroa vacciniforme includes a group of different observations which only in part correspond to cutaneous porphyria.

F. F. H.

KNUCKLE PADS (PULVILLUS DIGITI). J. RAMOS E. SILVA. (1956) *Ann. Derm. Syph., Paris*, **83**, 22.

Two cases are reported and attention is drawn with illustrations to their portrayal by famous artists notably Dürer and Michaelangelo, whose mighty David in Florence must have the biggest knuckle pads in the world. Histologically there is a dermal fibrosis and marked hyperkeratosis. The tendency to produce fibrous hyperplasia is more important than the actual provoking trauma.
F. F. H.

PROFUSE KERATOTIC LENTIGINOSIS. R. DEGOS and A. CARTEAUD. (1956) *Ann. Derm. Syph., Paris*, **83**, 125.

A girl aged 19 presented an explosive eruption of profuse dark lentigines especially on the thighs, arms and stomach. Many of them were covered by a thick squame. Histologically some lesions showed a typical lentigo covered with parakeratosis, others just a parakeratotic patch resembling ichthyotic scale.
F. F. H.

APHTHOSIS. HELEN OLLENDORFF CURTH. (1956) *Ann. Derm. Syph., Paris*, **83**, 130.

The author prefers this name to the more restricted one of Behçet's syndrome because its manifestations extend beyond the classical triad of uveitis, genital and buccal ulcerations and include skin manifestations such as erythema nodosum and lesions in the central nervous system which may even be fatal. The fundus oculi should always be examined as there may be changes in the retinal vessels. The primary lesion is a vasculitis with thrombosis and haemorrhage. The aetiology is unknown, the evidence of a virus being still incomplete. Treatment even with cortisone is unsatisfactory; the author favours blood transfusion.
F. F. H.

SCLERO-LICHENIFIED AND SCLERO-VITILIGINOUS LESIONS IN CUTANEOUS PORPHYRIA IN THE ADULT. M. BOLGERT, J. CANIVET and J. LÉPINE. (1956) *Ann. Derm., Syph., Paris*, **83**, 142.

Five patients are described with cutaneous porphyria, but no abdominal or nervous manifestations, who showed peculiar changes in the colour and consistency of the skin over the neck, face and upper chest. The skin in these areas showed red or whitish fairly well defined plaques with scleroderma-like lichenified or more rarely atrophic changes.
F. F. H.

ON ADIPONECROSIS SUBCUTANEA NEONATORUM. H. HERING and W. UNDEUTSCH. (1956) *Derm. Wschr.*, **134**, 917.

This condition which is hardly known to paediatricians is occasionally observed by dermatologists. The lesions occur in otherwise healthy infants and disappear spontaneously. Two cases are reported. One recovered completely after about 8 weeks, the other died having been removed from hospital against medical advice.

The lesions consisted of bluish red, apparently painless swellings which were found on the back in one, on the upper trunk and all extremities in the other.

Histologically there were characteristic interstitial haemorrhages in the fat tissue, coagulation necrosis and the presence of fatty acid crystals.

Mechanical trauma, cooling and increased sensitivity of the peripheral vascular system in infants are thought to be responsible for the condition. Contrary to what happens in the adult under similar conditions complete restitution takes place.
W. G. T.

SPECIAL LOCALISATION OF EPIDERMOPHYTON INFECTIONS. I. WAGNER. (1956) *Derm. Wschr.*, **134**, 885.

Increasing attention is drawn to infections due to *Epidermophyton interdigitale* and *Epidermophyton rubrum* causing clinical manifestations in atypical sites.

The clinical features do not usually suggest a ringworm aetiology and careful and often repeated search for the organism is indicated.

Of 33 cases the largest group consisted of follicular lesions occurring on the legs of women, often associated with keratosis pilaris and acrocyanosis. Vascular conditions such as thromboses and varicose veins as well as external factors e.g. occlusive dressings and elastic stockings are thought to predispose to the development of these atypical manifestations.
W. G. T.

ON THE MECHANISM OF URTICARIAL X-RAY REACTIONS. G. WEBER and O. BRAUN-FALCO. (1956) *Derm. Wschr.*, **134**, 892.

Treatment of a number of basal celled tumours with x-rays resulted in urticarial reactions at the sites within a few hours of exposure. Visible and ultraviolet rays did not produce this reaction in the patient. Passive transfer after Prausnitz-Küstner did not succeed. The fluid of cantharides blisters from the patient produced urticaria in experimental subjects after exposure to x-rays only. The wave length was approximately 0.25 A. W. G. T.

PATHOGENESIS OF THE FUNGUS FUSARIUM REDOLENS WR. (CLINICAL-EXPERIMENTAL INVESTIGATION). C. L. KOZIN. (1956) *Vestnik Ven. Derm., Moscow*, **1**, 28.

Fusarium graminearum contamination of grain is known to give rise to the peculiar intoxication caused by "tipsy bread", and the *Fusarium sporotrichioides* to an alimentary intoxication (septic angina) but the author could find in the literature no instance of pathogenicity in any strain of fusarium found in a skin lesion. In the author's patient, aged 43, with 2 painful chronic relapsing ulcers of the leg, 9×14 and 5×10 cm. *Fusarium redolens* WR. was repeatedly isolated side by side the microflora. The floor of the ulcer was uneven and necrotic and the edge uneven and in places undermined or raised and rolled, and cyanotic. The skin immediately adjacent was hot and of a bluish crimson colour. Autoinoculation with vaccine 1:10 and 1:100 caused hyperaemia and oedema up to 1.5 cm diameter and a positive response also with a 1:500 dilution of the culture (up to 1 cm. diameter). Controls (5 patients with gonorrhoea and one with epidermophytosis) with undiluted vaccine gave a negative result. The complement reaction (patient's serum and suspension of culture of the isolated strain) gave a brisk positive reaction: negative in the controls. Pathogenesis for rabbits and guinea pigs was proved. From the ulceration and necrosis following an abscess at the site of an intradermal inoculation in the rabbit, a retroculture was obtained and following inoculation of a guinea pig with a suspension, a culture of the fungus was grown.

The author describes in detail the appearance of the growth after 3 days on solid Sabouraud's medium and the findings in a hanging drop suspension after 1, 2 and 3 days, by now long branching mycelium and great quantities of spindles closely packed. (One clinical photograph and 3 photomicrographs.) M. S. S.

SOME LIVER FUNCTION TESTS IN PSORIASIS AND OTHER DERMATOSES. N. V. ILINA. (1956) *Vestnik Ven. Derm., Moscow*, **1**, 25.

The author used 3 methods of investigation:

- (1) the serum fasting urea and in the form of a curve after 3 gm of glycocoll.
- (2) total serum protein (refractometric test) and alb/glob ratio.
- (3) hippuric acid test for the antitoxic function.

A rise in the serum urea up to 50% in the 2-4 hours after glycocoll was considered a normal curve; above 50% a hyperfunctioning liver; absence of response—presence of liver dysfunction; a lowering of the urea content—severe dysfunction.

Of the 156 patients investigated, 74 had psoriasis, 42 eczema or dermatitis, 20 miscellaneous skin diseases and the 20 controls were practically healthy persons.

The controls (aged 23-46) gave an average fasting serum urea of 22.9 mg.% (15-33 mg.%); total protein 7.5 per 100 cc. (6.0-8.3) alb/glob index—2.0 (1.5-2.8).

In patients with widespread psoriasis: a lowered level of serum urea was found in the acute stage of the disease becoming normal in the retrogressing stage, e.g. pathological curves in the acute stage in 46%; retrogressing 33%; stationary stage 16%. To explain this persistence of 16% pathological curves it is suggested that the liver dysfunction is not dependent on the skin disease in these patients. Total proteins: in the progressive stage 6.2 per 100 cc.; stationary 7.3; retrogressing 7.5. Albumin/globulin ratio correspondingly: progressive stage 3.0; stationary 2.0; retrogressing 1.8. The antitoxic function of the liver: progressive stage 78; stationary 85%; retrogressing 83%. In patients with a localized form of psoriasis all deviation from the normal was considerably less marked even in the acute stage. In patients with eczema and dermatitis the level of serum fasting urea was more often raised than in acute psoriasis or in the controls. The level of urea did not change in dependence on the stage but in all there was a return towards the normal with recovery.

The author considers that the liver function tests could serve as prognostic signs and occasionally as a criterion for the effectiveness of therapy. M. S. S.